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TO THE ISSUE ON FETAL GROWTH RETARDATION PATHOGENESIS

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Fetal growth retardation (FGR) is one of the most acute medical problems connected to high number of perinatal diseases and deaths.
The objective: research of fetal growth retardation on basis of placenta's protein synthesis function and immunological parameters studying.
Methods. Î‘1-microglobulin (PP12), Î‘2microglobulin of fertility (PP14), trophic ?glycoprotein (SP1) and testosterone-estradiol-binding globulin (SSBG) were determined for evaluation of the protein-synthetic function of the placenta. To study immune responses, the indexes of activated lymphocytes content and macrophages in leukocyte infiltrate of the placental decidua were evaluated; cytokines production in placenta, synthesis of growth factors, parameters and regulation of immune apoptosis in placental decidua (sFasL, p53, Ð'Ñ•l – 2).
Patients: 157 pregnant women were examined at the gestational age of 24-42 weeks with early (67 women) and late (90 women) phenotype of fetal growth retardation. Results. Trophic ?glycoprotein (SP1), a specific marker of the placenta's fetal part, is synthesized by syncytio and cytotrophoblast cells. A decrease in this indicator was found in patients with FGR. PP12 is secreted by the decidua, being the marker of the maternal part of the placenta. The level of PP12 in pregnant women with an early form of FGR was considerably higher in comparison with the control (46.4 Â± 3.9 mg / ml, p <0.05), which may indicate destructive processes in the placenta. The study of the gene regulation of apoptosis showed that with FGR, sFasL and p53 expression by placenta cells is increased and Bsl-2 expression is reduced. The study of the cytokine profile of the placental decidua in pregnant women with FGR showed a decrease in IL-1Î², IL-8, IL10 and IFNÎ³, while IL2 and TNFÎ± were increased (p <0.05). Decreased production of cytokines and activation parameters of decidual macrophages may be the result of depression of protein-synthesizing function of the placenta in the pathological type of fetal adaptation.
In patients with an early form of FGR the level of TNFÎ±, enhancing the expression of integrins by the placental leukocytes, is increased, which can result in vascular thrombosis and ischemic necrosis.
Conclusions. In patients with FGR, placental protein-synthetic function disorder leads to a pathological orientation of the reactions of immunocompetent cells, placental destruction, prerequisites for vascular thrombosis and ischemic stroke.

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